REMARKS

Election

The undersigned confirms the election of Group I (Claims 1 and 2) and the election of species (a) and (b) of claim 2, without traverse. Claims 3-5 which stand withdrawn from consideration are canceled herewith, to advance the prosecution.

The Rejections

Claim 1 is rejected under 35 U.S.C. 112, first paragraph, for failing to comply with the written description requirement on the basis that the term "functionality" is not defined and on the basis that the specification does not teach all the COX-2 protein independent therapeutic activities that can be screened for.

Reconsideration is requested.

The term "functionality" is used in claim 1 with its normal dictionary meaning "of or able to perform a function". In any event, the functionality being screened for is made clear by the positive step recital.

So far as generically claiming is concerned, the recitations in claim 2 support the genus of claim 1. Moreover, the burden is on the PTO to establish that one skilled in the art would not know or be able to routinely establish other COX-2 protein independent therapeutic activity screens. The PTO has not met this burden.

Claim 2 (a) is rejected under 35 U.S.C. 112, first paragraph on the basis that COX-2 protein independent function is not being measured and because the meaning of "activation . . . by at least 100%" is unclear. Reconsideration is requested.

As indicated at page 4, luciferase activity indicates PPAR activation. A search on Google will indicate the therapeutic activity for PPAR activation. For example, known positives in the assay of 2(a) are provided by thiazolidinediones which are used to treat Type II diabetes. So far as "activation . . . by at least 100%" is concerned, the meaning is clear from page 3, three lines from the bottom.

Claim 1 is rejected under 35 U.S.C. 102(b) as anticipated by Vadlamudi. Reconsideration is requested. What is being tested is effect of NDF. NDF was found to induce activation of COX-2 promoter, expression of COX-2 mRNA, COX-2 protein, accumulation of prostaglandin E2, cell growth and cell invasion. See Abstract and Figure 9 at page 11. The inclusion of NS-398, a known selective inhibitor of COX-2, was found to inhibit production of prostaglandin E2, cell growth and cell invasion, leading the authors to suggest that these effects were COX-2 dependent. Consider the following quote from Vadlamudi:

> Abstract: ". . . Taken together, our findings provide the first biochemical evidence of a possible role of the COX-2 pathway in the mitogenic action of NDF . . ."

Thus, the testing suggests a COX-2 protein dependent effect. The point is that NS-398 was used in Vadlamudi, for its COX-2 inhibition function.

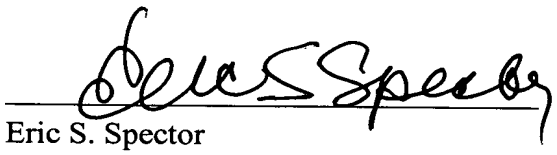
Claim 2 is rejected under 35 U.S.C. 103(a) as being obvious over Winde et al. in view of Subbaramaiah et al. Reconsideration is requested.

So far as Winde is concerned, sulindac is used and it is not a COX-2 inhibitor and even its metabolites are not selective inhibitors of COX-2 protein activity. Moreover, Winde does not describe a screen since testing was carried out eighteen months later on a human.

So far as Subbaramaiah et al. is concerned, it shows a COX-2 protein independent effect of thiazolidinediones and not of selective inhibitors of COX-2 and is therefore irrelevant.

Allowance is requested.

Respectfully submitted,



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